THE OCCURRENCE OF INFANTILE PARALYSIS IN MASSACHUSETTS IN 1907.

(With Especial Reference to Etiology.)

REPORTED FOR THE

MASSACHUSETTS STATE BOARD OF HEALTH.

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of Boston.

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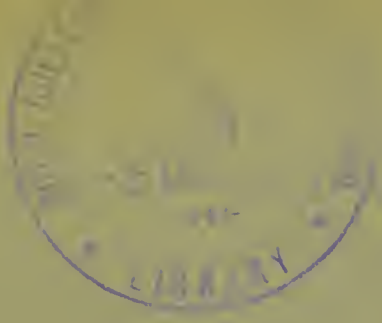


THE OCCURRENCE OF INFANTILE PARALYSIS IN MASSACHUSETTS IN 1907.

Infantile Paralysis (Anterior Poliomyelitis) is a fairly common disease which is followed by serious and most often disabling results in those affected. Its etiology is unsettled although it is generally considered as infectious in origin; it occurs in epidemics at times and is not free from the suspicion of being contagious. The epidemic of the disease which occurred in New York in the summer of 1907 was by far the most extensive ever reported in any part of the world, and left behind it hundreds of children crippled for life. Taking these facts into consideration, it seemed a suitable field of investigation by the Massachusetts State Board of Health in the interest of the public health, especially as it was reported to the Board by Dr. James J. Putnam that more cases than usual had been seen in Boston in the year 1907, although there was no evidence of any marked epidemic. The present report is to be regarded as a preliminary one, as the Board intends to pursue the subject further in the collection and analysis of future cases and in a bacteriological investigation of the disease already started by Dr. Theobald Smith.

In February, 1908, there were sent out to every physician in Massachusetts (some 6,000 in number) circulars asking them if in the year 1907 they had seen in their practice any cases of acute febrile disturbance followed by paralysis, especially in young children. To the physicians replying in the affirmative, blanks, following somewhat those of

the New York Committee, were sent to be filled out, one for each case, of which 234 have been received and analyzed. Although undoubtedly not all cases occurring in Massachusetts were reported, the data at hand may be taken as representative of conditions in the State, and it is fair to assume that most cases occurring find a place, and that the report may be taken as fairly representative of the occurrence of the disease in a State of 3,003,680 inhabitants living partly in cities and partly in the country.

The present report will deal with the data recorded in literature as to etiology, epidemic character, contagion, experimental production in animals, and then will be presented an analysis of the cases collected by the Board with especial reference to any light they may throw on the etiology of the affection or its clinical recognition. The group of cases presented differs somewhat from most material already collected, which is largely derived from the observation of late cases seen in hospital clinics, whereas these are acute cases reported by the attending physicians to whom the Board desires to express its obligation for their willing and efficient coöperation in the enquiry.

Proceeding first to the analysis of literature, the data are as follows:

BACTERIOLOGY.

The evidence in favor of the bacteriological source of the disease is as follows:

Scultze* in 1898 found a diplococcus in a lumbar puncture on the 13th day free in the spinal fluid; leucocytes were not present. A second puncture two days later was sterile.

Dercum† in a lumbar puncture found a micrococcus which in morphology and staining properties resembled the diplococcus of Sternberg.

* Münchener Med. Wch. 1898–38.

† Journal of Neu. and Ment. Dis. 1900–XXVII.

Spiller* found the staphylococcus pyogenes albus in cultures in the fluid from lumbar puncture in one case affected at the same time with small-pox.

Chapin† quotes Brooks as finding (1) a diplococcus in the anterior horns of the cord and not in the blood or meninges, and (2) a diplococcus in the blood during life.

Batten‡ found in the cerebro-spinal fluid (*a*) short difficult staining bacillus (probably proteus vulgaris), (*b*) a fat coccus like that from infected wounds, (*c*) the ordinary staphylococcus.

Huber§ on the third day identified in the fluid a diplococcus situated in the cells, typical of cerebro-spinal meningitis.

Engel‖ found staphylococcus albus, but the patient had also suppurating bone disease.

Barnes and Miller¶ identified the staphylococcus albus and pyogenes citreus in a case examined after death.

Bulow-Hansen and Harbitz** found in one case in a serumagar culture from the spinal fluid obtained post mortem a diplococcus or short double rod. Gram staining. This was not found in the sections of the cord; the organism was not virulent for animals, and seven other cultures were sterile.

Looft and Dethloff†† in two cases found a Gram-staining diplococcus which they identified with meningococcus type Heubner.

Geirsvold, Harbitz and Scheele‡‡ in 15 cases have found in the spinal fluid from lumbar puncture bean-shaped diplococci or tetrads growing in from 2 to 6 days on cultures.

* Brain. Autumn, 1903.
† Arch. of Pediatrics, Nov., 1900.
‡ Brain. 1904. 376.
§ Deutsch. Med. Wchsft. No. 12. '79.
‖ Prag. Med. Wchsft. 1900. No. 12. ¶ Brain. 1907.
** Norsk Mag. f. Läegevidenskaben. 1896. 11.
†† Medicinsk Revue. 18. 1901–321.
‡‡ Journal American Med. Assoc'n, Jan. 25, 1908.

They stained by Gram's method, except in old cultures where this was variable. They were virulent for animals, causing atrophy, paresis, emaciation and death.

Concetti* examined the fluid obtained by lumbar puncture in 9 cases of acute anterior poliomyelitis; in 2 cases the pneumococcus was present and in a third the meningococcus of Weichselbaum; the others were sterile.

Pasteur, Fullerton and Maccormac† found a diplococcus in the fluid withdrawn from an acute case which caused motor paralysis when inoculated into the subdural space of rabbits, but which could not be grown.

This represents all the positive evidence that it has been possible to find. A similar organism was found by Geiersvald in the throats of patients affected. The same coccus was, however, obtained from the throats of persons in localities where the disease had not appeared. On the other hand, most competent investigators have not found organisms in the spinal fluid during life or in the sections of the cord after death. Such findings are reported by Dauber, Goldscheider, Siemerling, Guinon Rist, and others.

Flexner‡ found that bacterial cultures both in aërobic and anaërobic media gave no results, and that the cerebro-spinal fluid was sterile, no inflammatory products being found in the fluid. The Mt. Sinai Hospital studies‡ showed only a moderate leucocytosis in a number of the cases, many bloods being normal.

Achard and Grenet,‡ in a fourteen-year-old child, found pronounced lymphocytosis in the cerebro-spinal fluid.

Frankel‡ and Niedner and Mamlock‡ found no such lymphocytosis.

That Anterior Poliomyelitis is an infectious disease is the commonly received opinion. Since it has been seen that it cannot be regarded as established by bacterial evi-

* Rev. mens. d. mal. de l'Infance. 1900, p. 550. † Lancet, Feb. 15, 1908.
‡ Journal American Medical Assoc'n, Oct. 19, 1907, p. 1370.

dence so far collected, the other evidence in favor of this view will, therefore, be investigated.

The disease appears to be a pathological entity, but it must be remembered that it is possible that it merely represents the reaction of the spinal cord to various causes, such as sepsis, trauma, specific infectious diseases such as measles, chilling of the body, and over-exertion. When the antecedents of the attacks are analyzed in this series of cases, it will be seen that these points must be considered.

EPIDEMIC CHARACTER.

That the disease is at times epidemic is unquestioned, and established by ample evidence; nor are epidemics confined to any especial part of the world, to any one kind of climate, nor, so far as can be seen, to any especial environment.

Holt and Bartlett* have collected thirty-five (35) epidemics, prior to 1907, recorded in literature, and have analyzed them and given the literature in full. The principal epidemics are as follows:

France, near Lyons. 13 cases in town of 1,500.†

Italy, near Florence. 7 cases in 15 days. Another epidemic, 17 cases in 4 months.‡ 2 epidemics in Conegliano of 9 and 13 cases (Fabis).§

Germany. In a polyclinic, averaging from 2 to 3 cases, yearly, between 1892–1897, between May and December, 1898, there were 15 cases.‖

Austria. 42 cases in and near Vienna in summer of 1898.¶

* American Journal Med. Sci. May, 1908. 647.

† Cordier. Lyon Med. 1887.

‡ Piericanni and Buccelli. Quoted by Johanessen.

§ Fabis La Pediatria. May, 1901.

‖ Jahrb. f. Nervenheilkunde. 1899, i, 41.

¶ Zappert Jahrb. f. Kde. 1901, 125.

Norway and Sweden. In Stockholm from 1888–1895 there were from 3 to 11 cases a year. In 1895, 21 cases from March to October, inclusive.*

Leegard†: July to Oct., 45 cases (preceded by colds in 6 cases, exanthemata in 7, overexertion in 9. An epidemic of jaundice existed in 2 of the districts).

Medin.‡ 2 epidemics in Stockholm in 1887 and 1895; 43 and 21 cases, respectively. (Cf. Johanessen for description of latter.)

Rissler.§ 5 epidemic cases with description of their pathology.

Harbitz and Scheele‖ dealt at length with the prevalence of the disease in Norway. In 1905, there were 719 cases with 34 deaths; in 1906, 334 cases with 34 deaths. Thus in the two years, 1,053 cases with 145 deaths (13.8% mortality). Most cases were typical anterior poliomyelitis, but cases were seen of acute ascending paralysis, and some encephalitis, but no clear cases of cerebro-spinal meningitis.

Other epidemics in Norway and Sweden were reported by Bergenholz in Umea, in July, 1881.¶

Australia. Alston** reported an epidemic of 14 cases in Fort Lincoln, South Australia, in March and April, 1895.

Wade†† reported an epidemic of 34 cases where pain was a prominent symptom, occurring in 28 of the 34 cases.

United States. Epidemics from widely separated parts of the country have been reported.

* Johanessen. Festschft of Abraham Jacobi. 1900, p. 263.
† Leegard. Abst. in Neurol. Centralbl. 1902. xxi-505.
‡ Medin. Nord. Med. Arkiv. 1896.
§ Rissler. Nord. Med. Arkiv. 1888–22.
‖ Journal American Med. Assoc'n. Oct. 26, 1907, p. 1420.
¶ Medin. Nord. Med. Arkiv. 1896.
** Alston. Australasian Med. Gaz. April 24, 1897. 123.
†† Wade. Australasian Med. Gaz. July 24, 1904.

Newmark* reported 4 cases in a village of 49 inhabitants near San Francisco.

Bondurant and Woods† described 15 cases occurring in Alabama within a radius of 12 miles.

Colmer‡ reported an epidemic in West Feliciana, Louisiana.

Brackett§ investigated 10 cases occurring in North Adams, Mass., in 1894. (As a rule the cases were situated along one or two rivers flowing through the town. The bladder and rectum were affected in an unduly large proportion of the cases.)

J. M. Taylor‖ reported 7 cases occurring in Cherryfield, Maine.

Painter¶ described an epidemic in Gloucester, Mass., of 38 cases with one death. No common etiological factor was found.

An epidemic in Poughkeepsie, New York, was observed in the summer of 1901 by H. L. Taylor, of New York.**

One of the most extensive epidemics ever reported occurred in Rutland, Vermont, and was investigated by Caverly†† and McPhail,‡‡ where there occurred 132 cases in the summer of 1894 with 18 deaths. There was nothing in the climate, soil, locality, or class affected, to explain the distribution.

In New York city, in the summer of 1907, occurred the most severe and extensive epidemic ever reported.

A fact perhaps bearing on the etiology is found in the

* Medical News. Jan. 28, 1899.

† Bondurant. Medical News. Aug. 18, 1900.

‡ American Journal Med. Sci. 1843.

§ Trans. Am. Orth. Assoc'n. vol. xi, p. 132.

‖ J. M. Taylor. Phila. Med. Journal. Jan. 29, 1898.

¶ Boston Med. and Surg. Journal. Dec. 11, 1892, p. 633.

** Quoted by Painter.

†† N. Y. Med. Journal. 1894. Vol. 2.

‡‡ Brit. Med. Journal. 1894. Vol. 2, p. 1233.

affection of five dogs on the Labrador coast* in the autumn of 1907. The dogs were feeding on decomposed herring which was full of maggots, and were affected by a sudden paralysis, most marked in the hind legs; three died and two recovered. No post mortem examination was made.

These data establish the fact that the disease is at times clearly epidemic, but from these epidemics no definite data have been obtained to explain the etiology of the disease or the mode of infection. The statement is made by Leegard that in the epidemic observed by him the disease spread along the lines of most extensive travel. The statement is made by Norwegian authors that localities visited by an epidemic were comparatively immune for awhile.

CONTAGION.

Evidences of contagion or of a source of common infection, as from a milk supply, are not lacking and deserve consideration.

The conclusion of Geirsvold,† who investigated the Swedish epidemics, was that the disease was contagious, frequently several cases occurring in one family. This conclusion was concurred in by Harbitz and Scheele.

Holt and Bartlett, in their analysis of literature, found 40 instances in which more than one case of the disease occurred in a family or household. These were tabulated as follows:

Two cases in a family, 31 instances.
Three cases in a family, 5 instances.
Four cases in a family, 3 instances.
Seven‡ cases in a family, 1 instance.

* Personal communication by Dr. H. M. Hare of Harrington Hospital.

† Quoted by Harbitz and Scheele.

‡ Pasteur. Trans. Clin. Soc. of London. 1896; 143.
Buzzard. Lancet, 1907; pp. 705, 785, 865.

The intervals between the illness of the different patients were as follows:

Same day, or simultaneously,	8 instances.
Interval one day,	2 "
" two days,	1 instance.
" three days,	1 "
" four days,	5 instances.
" five "	2 "
" six "	2 "
" seven "	5 "
" eight "	2 "
" six weeks,	1 instance.

EXPERIMENTAL EVIDENCE.

As bearing on the fact that the disease may be of multifold origin, the following facts must be considered.

Stieglitz* experimented with lead poisoning in 36 animals and in one guinea-pig found characteristic changes with destruction of cells in the anterior cornua with cell infiltration. Paralysis was present and death occurred in 24 hours.

Vulpian † produced, experimentally, paralysis of the extensors and lesions resembling those of poliomyelitis in a dog by lead poisoning, and in a case of lead poisoning found pronounced poliomyelitis with colloid degeneration and cell atrophy.

Phillippe and Gauthard ‡ report a case of anterior poliomyelitis from lead poisoning, and Obrastoff § one from arsenical poisoning. Onuf ‖ reported the case of a painter with flaccid paralysis of both legs, in whom autopsy showed lesions characteristic of the disease.

Turning from this to experimental data bearing on the

* Journal of Nerv. and Ment. Dis. 1900. Vol. 27, p. 156.

† Maladies du Syst. Nerveuse. Paris. 1879.

‡ Neurol. Centralblt. 1903. XXXI-889.

§ Neurol. Centralblt. 1902 XXII-278.

‖ Journal of Nerv. and Mental Dis. 1900. XXVII. 155.

subject, it is recognized in laboratories that paralysis occurs at times in young rabbits not experimented on. It has been described in guinea-pigs* after the injection of toxin which has been only partially neutralized by antitoxin and very rarely after the injection of toxin alone, and in animals, especially rabbits, made septic, it frequently is seen.

Too much weight, therefore, cannot be attached to the following suggestive experiments.

Thoinot and Masselin † injected 43 rabbits in the marginal veins of the ear with a culture of the colon bacillus: 9 died of sub-acute general infection and lesions were found in the anterior cornua of the cord; 34 of the rabbits survived, and were all paralyzed in a period varying from 2 days to 6 months. The paralysis affected first and most the posterior limbs, extending in severe cases to the anterior. The paralysis at times was unilateral in its predominance. Diarrhœa was noted in all but three subjects, but the paralysis always recovered if the animal lived. In the cord congestive and hemorrhagic phenomena were noted with degeneration of the cells in the anterior cornua of the cord, the peripheral nerves were normal and the muscles atrophied. The same phenomena appeared in all but one of six survivors of a similar injection of a culture of the staphylococcus aureus in nineteen rabbits, of which eleven died. Similar phenomena were reported by Gibert and Lion ‡ from injection of the colon bacillus; by Bourges, from injection with the coccus of erysipelas, and by Vincent from injection of Eberth's and another bacillus, and by Charrin and Claude § by a pyocyaneus toxin.

Roger ‖ produced in rabbits a weakness of the limbs and muscular atrophy by the injection of a streptococcus of

* Lewis, Journ. Med. Research. XV-3.

† Revue de Med. 1894. 14. 449.

‡ Societé de Biol. 1892. Feb. 13.

§ Comptes Rend. de l'Acad. des Sciences. 1897–1133.

‖ Ann. de l'Inst. Pasteur. 1892. VI. 437.

moderate virulence with degeneration of cells in the anterior cornua.

Hoche* injected into the lumbar arteries of dogs aseptic pollen and other finely divided granules, with the result of producing in the spinal cord lesions much like those of infantile paralysis in certain respects, embolic softening or infarcts resulting.

It would seem justifiable to sum up the evidence as follows: The positive bacterial findings are not convincing, only a few such being reported and these not always agreeing as to the organism, whereas a very great number of cases are examined by lumbar puncture with negative results. But negative results do not disprove a bacterial origin for the disease, for the organisms may do their work and disappear or the harm may be done by toxines and not by the bacteria themselves. If one cannot accept the positive evidence as convincing, one must also remember the true value of the negative evidence in this connection. The injection experiments do prove, however, that certain metallic poisons, bacteria and toxines have a selective action on the motor cells of the anterior cornua when present in the general circulation, that the paralysis of this type may be largely unilateral, that the posterior limbs are always more affected than the anterior, and that the lesions in the cord in such cases do not differ essentially from those of anterior poliomyelitis.

The data from the study of the 234 cases in this series will next be taken up for consideration.

DISTRIBUTION.

It became evident on grouping the cases that certain foci at widely separated parts of the State had existed. One occurred in Pittsfield, in the western part of the State, a city of 25,700 inhabitants (with 18 cases), and north and

* Arch. f. Psych. 1899. XXXII. 209.

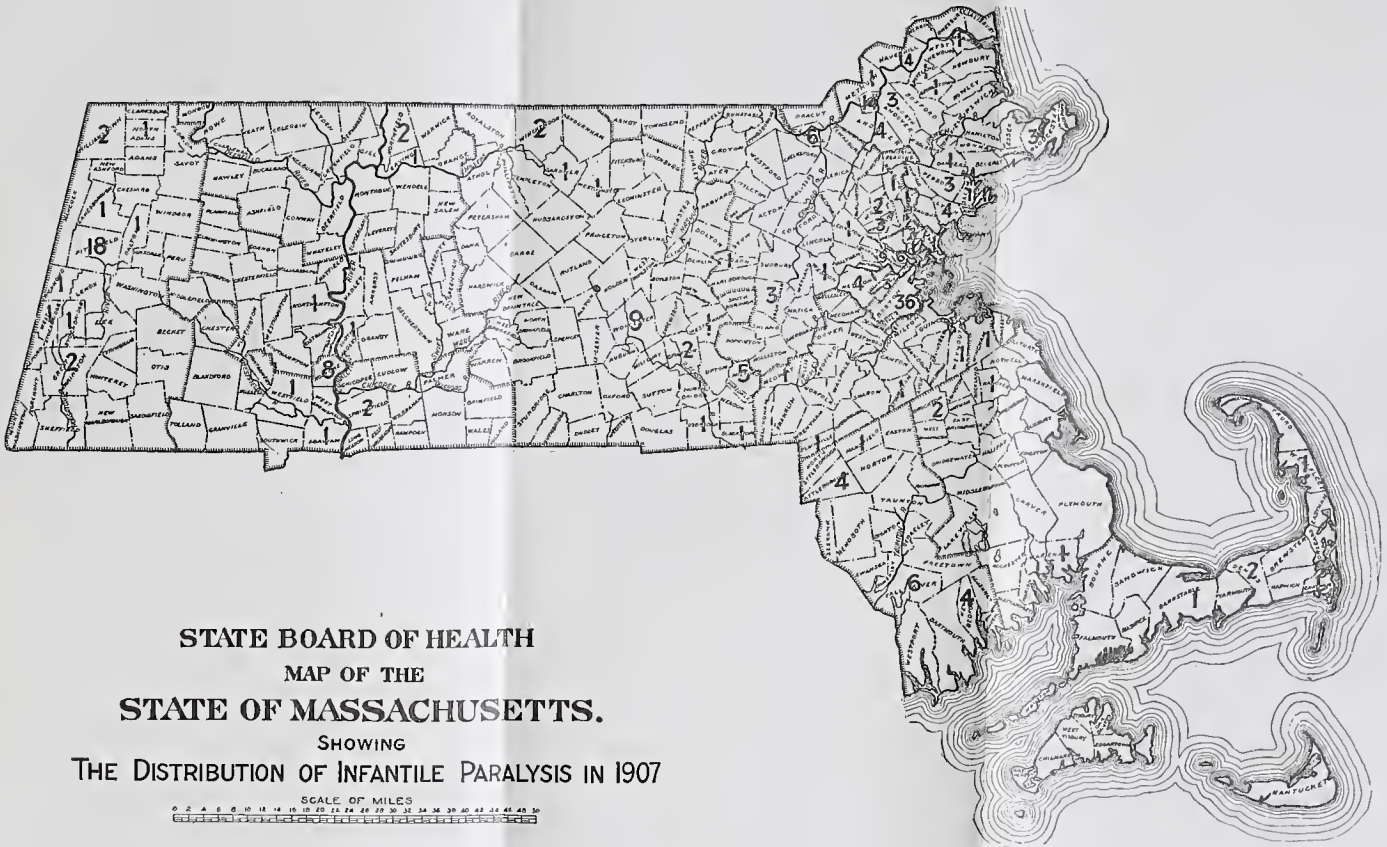
south of Pittsfield, along the lines of trolley travel, which is here extensive, appeared other cases in the adjoining towns. It did not spread east and west, in which directions the trolleys do not run. Of course, the trolley lines run in the most thickly populated regions, and the disease would naturally spread in the directions where most people were to be found. Still, it may be said, as in a Swedish epidemic, that the spread of the disease followed the lines of most extensive travel. There were no other cases reported in the western part of the State except those directly adjacent to Pittsfield. Coming east through an agricultural country, there were no more cases reported until the valley of the Connecticut River was reached, where there were a few cases along the valley in the adjacent cities of Springfield, Holyoke, and Northampton and South Hadley Falls, where in an aggregate population of about 127,000 there were 12 cases.

Another obvious centre of the disease was to be found in the group of adjacent towns in the valley of the Merrimac River, consisting of Lawrence, Haverhill, Georgetown and Andover, where, in an aggregate population of about 117,000, there were 36 cases, a prevalence much less than in Pittsfield, but much more than in the Connecticut Valley.

Boston, with 609,761 inhabitants, reported only 36 cases, and the remainder of the cases were to be found in various parts of the State, mostly in the eastern part, but practically always in contiguous towns; that is, it was rare to find even one case in a town without finding a case in one or more adjacent towns.

Distribution of cases of infantile paralysis in Massachusetts in 1907.

Agawam		1	Blackstone		1	Roxbury	7	
Andover	3		Boston	15		S. Boston	5	
W. Andover	1		Charlestown	1			—	36
	—	4	Dorchester	5		Brockton		2
Attleborough		4	E. Boston	2		Brookline		3
Barnstable		1	Jam. Plain	1		Cambridge		9



STATE BOARD OF HEALTH

MAP OF THE

STATE OF MASSACHUSETTS.

SHOWING

THE DISTRIBUTION OF INFANTILE PARALYSIS IN 1907

SCALE OF MILES

0 2 4 6 8 10 12 14 16 18 20 22 24 26 28 30 32 34 36 38 40 42 44 46 48 50

Chatham (West)	1	Lanesborough	1	Richmond	1
Chelsea	1	Lawrence	14	Salem	1
Dalton	1	Lexington	1	Somerville	4
Danvers	1	Lowell	6	W. Somerville	2
Dennis	2	Lynn	4	—	6
Erving	1	Malden	3	Springfield	2
Fall River	6	Mansfield	1	So. Hadley Falls	1
(Case No. 74, Tiverton, R.I.)		Marblehead	1	Stockbridge	1
Framingham	1	Medway	1	Stoughton	1
S. Framingham	2	Melrose	2	Uxbridge	1
—	3	Methuen	1	Wakefield	1
Gardner	1	Milford	5	Wareham	1
Georgetown	1	New Bedford	4	Wellfleet	1
Gloucester	3	Newburyport	1	Westborough	1
Grafton	1	Newton	1	Westfield	1
N. Grafton	1	Chestnut Hill	2	Westminster	1
—	2	N. Upper Falls	1	Weston	1
Great Barrington	2	—	4	West Stockbridge	1
Groveland	1	North Adams	1	Weymouth (East)	1
Hanover (West)	1	North Andover	3	Williamstown	2
Haverhill	12	Northfield (East)	2	Winchendon	2
Bradford	2	Northampton	1	Winchester	3
—	14	Peabody	3	Winthrop	1
Hingham	1	Pittsfield	18	Woburn	2
Holyoke	8	Plainville	1	Worcester	9
Hudson	1	Reading	1		
		Revere	1		

56 of the 234 cases (24%) occurred in towns of less than 10,000 inhabitants, and 25% of the inhabitants of the State live in towns of less than 10,000 inhabitants. The disease cannot therefore from these figures be regarded as attacking especially the inhabitants of the cities.

This evidence tends rather towards supporting the contagious character of the disease, as established by its uneven distribution, extending from foci and not evenly scattered through the State; by its extension from Pittsfield along the lines of most frequent travel, and by the fact that there was rarely a case in one town without the occurrence of a case in the adjacent town or towns.

Following up still further the evidence of contagion: other cases in the family were reported in 11 instances; other cases in the same house in 9 other instances; other cases among acquaintances in 20 instances. That is, in 40

cases (17%) there was reason to look into the question of contagion.

The histories pointing to contagion were then analyzed. A child of 3½ was affected, followed 11 days later by the father (reported by Dr. Pitcher of Haverhill). Two brothers were affected at an interval of two days (Dr. Hubbard of Holyoke). Two brothers were affected with an interval of two days (Dr. Philbrick of Northfield). Two brothers were affected, interval not stated (Memorial Hospital, Worcester). Two brothers were affected at an interval of ten days (Dr. Boland of South Boston).

Going outside of the family there were other suggestions to the same effect. Dr. Hubbard of Holyoke reported one case followed by another six days later in a playmate. Dr. Croston of Haverhill reported a case which was followed by the similar affection in a playmate five days later. Dr. Hayes of South Boston reported two cases occurring in playmates "at about the same time." Dr. Kelley of North Attleboro' reported a similar case in two children living opposite each other on the same street. In seven cases the statement was simply made that similar cases had occurred among patients' acquaintances.

In one instance a patient in the second house from the patient was affected, and in another there were said to be two other cases on the same street.

The inquiry as to other illness in the family at the time brought out the fact that in several instances febrile disturbances existed in other children in the same families, a matter which is of interest and has been noted in certain epidemics reported. Such febrile attacks have been regarded as abortive cases (Bulow-Hansen, Buzzard, Harbitz and Pasteur).

TRAUMATISM.

The frequency with which a traumatic history was given as preceding the attack was then analyzed. In 52 of the

234 cases such a history was given. A further analysis showed that in 3 of these cases the stumbling was probably connected with the attack. In 5 the history was not satisfactory or was too indefinite. In 9 the accident had occurred a month or more before the attack. Throwing out these cases, there remained 35 cases where a clear history of accident preceded the attack, generally within a few days of the onset. These accidents were both slight and severe; falling out of bed or from a chair, falling from a bicycle or wagon, blows on the head, falling down stairs, etc., formed the bulk of the histories, but in most instances where it was mentioned it was stated that the child fell on the head.

Skeptical as one may be about the value of traumatic histories, it must be remembered that these histories are not from the hospital class, that they are obtained from the family physicians who have reported the cases individually, and that they must be allowed considerable weight.

In 2 cases the child received a severe fright before the attack. In 4 a chill from falling into water was attributed as the cause. In 3 over exertion was thought to have been the cause.

REASON OF ONSET.

The disease is well recognized as one that attacks children in the first dentition, and as one that prevails in the late summer and early fall. In this it offers a striking resemblance to the prevalence of the gastro-intestinal diseases of children which affect children of the same age at much the same time of year.

As to age, the largest number of cases occurred between 1 and 2 as is always the case, 47 cases occurring in this year, and from then on the number of cases fell with more or less regularity to reach a level of from 1 to 3 each year after puberty was reached. There were 131 males and 103 females affected.

The greatest prevalence of the disease in these cases was in September, and the least from January to June inclusive. From July it rose to reach its height in September, and fell through October, November and December.

MONTH OF ONSET OF INFANTILE PARALYSIS.

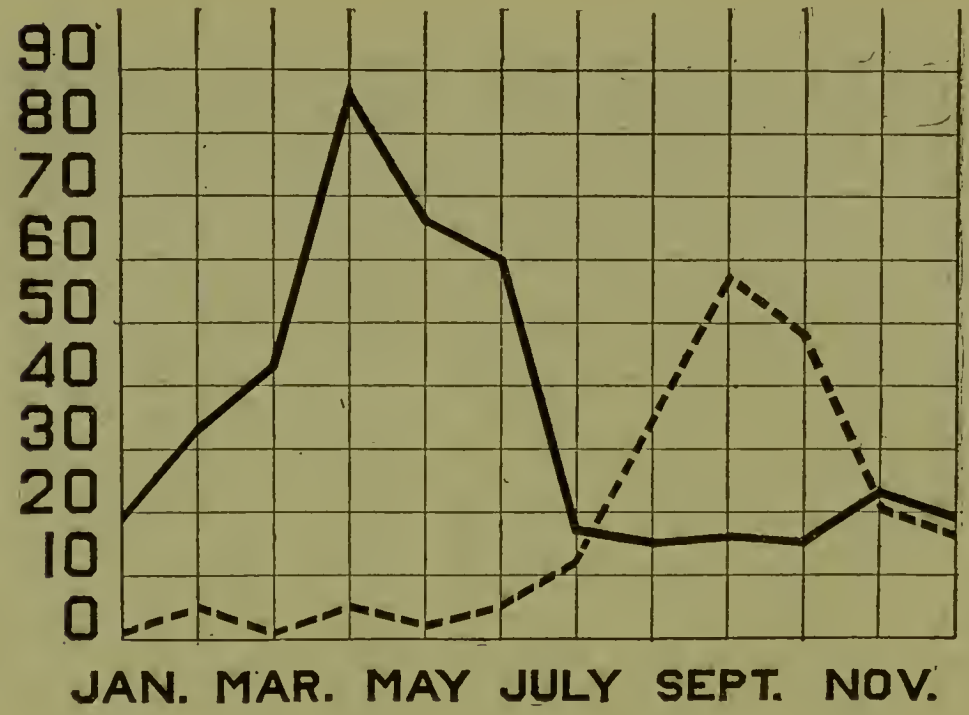
January	1	September	57
February	5	October	48
March	1	November	21
April	5	December	16
May	2	Not stated	29
June	4		
July	12		234
August	33		

OF CEREBRO-SPINAL PARALYSIS.

January	19	July	17
February	33	August	15
March	43	September	16
April	86	October	15
May	66	November	23
June	60	December	19

The cases of cerebro-spinal meningitis occurring in Massachusetts in the year 1907 (412 in number) were then analyzed to see how the seasonal occurrence of that disease corresponded to the prevalence of infantile paralysis. In cerebro-spinal meningitis the largest number of cases occurred in April, and the smallest number in August, September and October. As the relation of the two diseases has been discussed of late, some authors regarding them as practically variations of the same essential disease, it may be said that if the two have a relation of seasonal occurrence it is that infantile paralysis largely replaces cerebro-spinal meningitis during certain months.

The close correspondence between the age and season of occurrence of this affection, and that of the gastro-intestinal diseases, suggests the intestines as a source of infection possible from some bacillus contained in milk. In 201 instances where the question was answered, in 152 the patient was a



DISTRIBUTION OF 234 CASES OF INFANTILE PARALYSIS ------
" " 412 " " CEREBRO-SPINAL MENINGITIS } ——
BY MONTHS (MEAN IN 1907)

consumer of raw cow's milk and in 49 was not. That is, two-thirds of the patients reported on might have received their infection in this way, and the others undoubtedly were to some extent consumers of milk; on the other hand, it must be remembered that Geirsvold found in the throats of patients with infantile paralysis, a diplococcus resembling that found in the spinal fluid, but in this connection it must also be remembered that this is not an unusual state of affairs in patients without anterior poliomyelitis.

Taking up other factors in the history bearing on etiology, investigation as to race and nativity showed nothing of importance.

The reports on the conditions of the house in which they lived are of interest. 115 lived in detached houses; 110 in tenements.

As to the influence of dampness, 99 lived on the first floor, 65 on the second, 12 on the third, and 6 in the upper stories; 20 occupied the whole house.

Sanitary conditions were described as exceptionally good in 21. Good, in 123. Fair, in 55. Poor or bad, in 23.

The location of the dwelling was given as high and dry in 141. Damp or low or both in 76. "Medium" in 7.

Mosquitoes were reported as prevalent in 57 cases, and not prevalent in 156. Flies were prevalent in 90, not prevalent in 116. The house was screened in 133 cases, and not screened or insufficiently so in 80.

Analyzing these data for what they are worth, it would seem that it was not an affection confined to the lower classes, as shown by the simple fact that 133 out of 212 cases reported had their windows screened, which is an evidence of a fair amount of comfort. It did not affect the dwellers in tenements as often as it did those living in detached houses. That dampness may be a factor of possible importance is indicated by the fact that 99 out of 202 reported on lived on the lower floor, and that in 76 out of 224

cases the dwelling was low or damp. It would not seem probable that 49 per cent. of the population of the State lived on the lower floor or that 34 per cent. of the population lived in low or damp localities. It would seem, therefore, fair to assume that dampness was possibly a predisposing factor.

An enquiry as to whether any special disease was prevalent at the time in the locality elicited nothing of importance, and no instances of disease in the domestic animals of the family were found, except in one case—distemper in a dog.

CONDITIONS PRECEDING ONSET.

Various degrees of illness preceded the attack, malaise, headache and loss of appetite being the most frequent signs. This frequently preceded the attack for two or three days. The exanthemata had apparently some influence. Twice varicella occurred just before the onset, twice the paralysis occurred in the convalescence from measles, and once a rash thought to be scarlet fever preceded the attack. The presence of a possible source of septic absorption was shown once in the existence of an abscess behind the ear, once in chronic otorrhoea, once in a child vaccinated two weeks and once in a child vaccinated three weeks previously.

The degree of fever during the onset was as follows:

DEGREE OF ACCOMPANYING FEVER.

No fever	12	
"Fever"	75	(High, 8; moderate, 8.)
Temp. 100	6	
" 101	15	
" 102	33	
" 103	28	
" 104	14	
" 105	6	
Not noted	3	

Other symptoms in the attack were as follows:

SYMPTOMS ACCOMPANYING ATTACK.

	Present.	*Absent.*	*Not Noted.*
Vomiting	125	64	45
Brain symptoms	54	65	115
Retraction of head	64	84	86
Pain or tenderness, during or after attack	126	78	30
Digestive disorders	80	47	107
Disturbance of bladder function	42	158	34
Disturbance of function of rectum	44	152	38

The frequent occurrence of pain and sensitiveness during or after the acute attack is emphasized in this series, and is not sufficiently recognized in general frequently leading to errors in diagnosis. That it was present in 61% of all cases noted means that it is a symptom to be expected and remembered.

The duration of the acute attack was as follows:

DURATION OF ACUTE ATTACK.

1 day or less	10 cases	6 days	9 cases
2 days	19 "	1 week	21 "
3 days	23 "	1–2 weeks	33 "
4 days	17 "	2–3 weeks	23 "
5 days	13 "		

(A few longer durations are given.)

RELATION OF BEGINNING OF PARALYSIS TO ONSET OF FEVER.

Paralysis preceded the attack in 2 cases.

It occurred immediately after in	30 cases
within 12 hours	10 "
within 24 hours	20 "
within 48 hours	25 "
within 3 days	28 "
within 4 days	21 "
In from 4 to 7 days	34 "

(A few longer intervals are given.)

DISTRIBUTION OF PARALYSIS.

The distribution of the paralysis when at its worst was as follows:

		Late cases from Children's Hospital Series.
R. Lower Extremity	30	216
L. Lower Extremity	31	239
Both Lower Extremities	41	130
R. Arm	3	5
L. Arm	14	5
Both Arms	3	0
R. Arm, L. Leg	8	5
R. Arm, R. Leg	11	7
L. Arm, L. Leg	16	8
L. Arm, R. Leg	1	2
Both Arms, R. Leg	0	0
Both Arms, L. Leg	2	0
R. Arm, Both Legs	4	1
L. Arm, Both Legs	11	1
Both Arms, Both Legs	23	3
Back (In connection with other Paralysis)	40	—
Abdomen (In connection with other Paralysis)	19	—
Face (In connection with other Paralysis)	15	—
Not noted	28	

For purposes of comparison a table is given of 635 late cases reported from the orthopedic clinic of the Children's Hospital, Boston,* showing how much less extensive is the late distribution of the paralysis than is the early distribution. It is a matter of common information that a period of spontaneous improvement follows almost directly on the onset. This period lasts from two months upward, during which the paralysis limits itself most often to one or two members, generally to the lower extremity.

It is to be regretted that in the present series of cases the notes of the final condition were not sufficiently full to enable us to make a table of the end results.

A fact of interest, not recognized at any rate by the orthopedic surgeons, who from seeing late cases have come to

* Report not yet published.

the conclusion that permanent and complete recovery from the disease is at least very rare, is found in this series of cases which indicate that complete recovery is not unusual and 18 well authenticated cases of what appears to have been complete recovery of muscular power have been reported.

The paralysis in these cases was located as follows:

Both lower extremities,	4
One lower extremity,	5
Arm and leg, same side,	2
Hemiplegic distribution,	2
One arm,	3
General slight paralysis,	2

The time given for recovery varied quite evenly from 2–12 weeks, but two cases of recovery in from 24–48 hours were given. These were as follows:

Dr. Palmer, of South Framingham, reported a case six months old, where a total paralysis of the left arm followed 24 hours after an attack of fever. The loss of power lasted 24 hours and wholly disappeared.

Dr. Philbrick, of East Northfield, observed a child three years old attacked by fever of 101, retraction of the head, and twitching of the face, and the attack was followed immediately by some paralysis of both arms and legs. Recovery followed in 48 hours. The treatment consisted of free catharsis.

The tabulation of the time of recovery was as follows:

24 hours	1	2 months	3
24–48 hours	1	2½ months	2
2 weeks	2	3 months	3
3 weeks	1	Gradual	1
1 month	2		
6 weeks	1	Total	18
7 weeks	1		

There seems no reason to doubt the authenticity of these cases; on the whole the histories suggest that they were to be classed among the less severe cases, but such

histories as the following were not unusual. Case No. 105. (Dr. Hubbard, of Holyoke.) Temperature, 103. Vomiting. Brain symptoms. Retraction of the head. Pain in the left leg. Digestive disturbance. Paralysis of the left leg and thigh. Complete recovery in six weeks.

If such cases as this are not true anterior poliomyelitis, they certainly resemble it sufficiently to warrant the statement that cases of considerable severity, not to be distinguished from anterior poliomyelitis, may recover entirely or apparently so in a few weeks, a course quite in accord with the experimental paralysis produced in animals above alluded to.

Death occurred in eleven cases; once there was a complicating pneumonia, four times the fatal result was apparently due to an involvement of the respiratory muscles by the paralysis, once was death preceded by a temperature of 105 and delirium. There was no information obtained in any case by autopsy.

The examination of the urine showed nothing of importance, 63 out of 66 urines examined being reported as normal or showing only evidences of febrile disturbance, while three contained albumen.

The Examination of the Blood was reported in eleven cases, nine being normal, one showing a "low color index" and one having a leucocytosis associated with a complicating lobar pneumonia.

SUMMARY.

The evidence as to the etiology of the disease may be summed up as follows: The sudden febrile onset and the character of the onset suggest infection, and there is a general impression that the disease is infectious, but the case is not proved by positive bacteriological data so far collected and most bacteriological examinations are negative, But

in this case negative evidence does not disprove the theory of infection, for the organism present, if one exists, may liberate a toxine and disappear.

The seasonal occurrence, the age of the children selected most commonly, and the frequent association of intestinal disturbance with the onset, suggest some intestinal infection as the possible source of the disturbance. It must be remembered that the disease follows the curve of summer diarrhœa as to age and season and follows the curve of cerebro-spinal meningitis as to neither.

From the evidence at our disposal it seems reasonable to suspect that some bacillus, probably an anaërobic one, reaches the intestines in milk and there liberates a toxine which is absorbed and carried to the spinal cells by the blood current. Hence the findings in the cerebro-spinal fluid withdrawn by lumbar puncture are negative in most cases.

But it cannot be regarded as certain that infantile paralysis is always caused by the same organism nor even that it is a pathological entity. It may be simply the clinical expression of the reaction of the spinal cord to one of several causes of which infection may well be one. A similar pathological condition has been seen to arise from lead poisoning from the experimental injection of bacteria in rabbits and from the injection of toxin in guinea pigs. Allowance must also be made for the possible influence, as shown in this report, of traumatism, dampness, over fatigue, the exanthemata and foci of pyogenic infection, as possible causes of infantile paralysis or a disease indistinguishable from it. At present we must observe and study and collect material, remembering that we may be dealing (1) with a specific infectious disease, (2) with an infection due to one of several organisms, or (3) with a disease of more than one origin not always necessarily infectious.

As to contagion, the data contained in this report are

not sufficient to establish this characteristic, although the distribution of the disease, its spread from foci, the involvement of contiguous towns, the spread along lines of most frequent travel, and the very suggestive histories given here, may well warrant us in suspecting it and collecting further data, and no harm could arise from the isolation of such cases from other children during the acute attack.